THE INTERPRETATION OF THE VENOUS PULSE.[1]

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The knowledge of the existence of pulsations in the veins dates back to 1704 when Homberg[2] presented to the French Academy the result of his observations on the venous pulse. Morgagni,[3] however, was the first to offer rational views concerning this phenomenon. His dissertation upon the venous pulse is to be found in his work, *De sedibis et causis morborum*, which appeared in Venice in 1762. The first attempts to study the venous pulse experimentally were made by Barry[4] in 1826 and by Wedemeyer[5] in 1828, but owing to the inadequate methods used, their results were rather unsatisfactory. Bamberger[6] in 1856, and others following him, in common with their clinical predecessors, described the venous pulse as occurring only in tricuspid insufficiency. Bamberger, later (1863) published tracings of the venous pulse of tricuspid regurgitation, while Marey[7] in the same year took graphic records from animals. Friedreich,[8] in 1865, showed its presence in conditions other than tricuspid insufficiency, and described it as being formed of two waves: one presystolic in time and due to the contraction of the auricle, the other systolic in time and due to the shock of the aorta against the superior vena cava. Potain,[9] in 1868, described the venous pulse as composed essentially of a positive wave, due to the auricular systole, followed by two negative waves, the first being caused by the auricular, the second by the ventricular diastole. The same observer, in 1881, published tracings taken simultaneously from the jugular vein, and either the carotid artery or the cardiac apex. In the same year Riegel[10] interpreted the normal venous pulse very much as Friedreich had before him, believing, however, that the principal wave was that originating in the contraction of the auricle.

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[1] Read by invitation at a meeting of the Pathological Society of Philadelphia, April 9, 1908.

[2] Sur un battement de veines semblable au battement des artères. Hist. acad. roy. d. sc., Paris, 1704. Amst. 1707, 218 to 223. Also, Rec. d. mem., Dijon, 1754, ii, 138 to 140.

[3] De sedibis et causis morborum, Venice, 1762. Letter XVIII, Sec. 9, 10, 11.

[4] Recherches expérimentales sur les causes du mouvement du sang dans les veines. Mem. Acad. d. Sc., Paris, June 8, 1825. Paris, chez Crevot, 1825. Experimental researches on the Influence of Atmospherical Pressure upon the Progression of the Blood in the Veins, etc., London, T. and G. Underwood, 1826.

[5] Untersuchungen über den Kreislauf des Blutes und insbesondere über die Bewegung desselben in den Arterien und Capillargefässen, Hanover, 1828.

[6] Lehrbuch der Krankheiten des Herzens, 1857. Beobachtungen über den Venenpuls. Würzb. med. Ztschr., 1863, iv, 232.

[7] Physiologie méd. de la circulation du sang, 1863.

[8] Ueber den Venenpuls. Deut. Arch. f klin. Med., 1865 to 1866, i, 241.

[9] Des mouvements et des bruits qui se passent dans les veines jugulaires Mem. soc. med. hop. de Paris, 1867 and 1868.

[10] Ueber den normalen u. pathologischen Venenpuls. Deut. Arch. f. klin. Med., 1882, xxxi, 1. Experiment. Untersuchungen über den normalen Venenpuls, Deut. Arch. f. klin. Med., xxxi, 470.

Since that time most of the descriptions given in text-books on diagnosis, have embraced two forms of venous pulse: (1) The physiological venous pulse, characterized by a presystolic positive wave attributed to the arrest or slowing of the flow of the blood in the veins as a result of the auricular contraction; since this wave is immediately followed by a negative wave synchronous with the arterial pulse, this form of venous pulse is sometimes called systolic venous collapse; (2) the pathological or centrifugal regurgitant venous pulse consisting of a systolic positive wave due to a regurgitation of the blood into the auricle, and, possibly, also into the veins during ventricular systole and characteristic of tricuspid insufficiency, whether it be organic or functional.

François Franck[11] and later Fredericq,[12] showed that in the absence of tricuspid regurgitation a slight systolic wave is constantly present in the jugular veins of animals. Franck attributed the formation of this wave to the shock of closure of the auriculoventricular valves, while Fredericq believed it to be due to the sudden projection of these valves into the auricle at the beginning of ventricular systole. They both interpreted the presystolic wave as their predecessors had: as caused by the auricular systole. They also called attention to a third wave occurring at the end of ventricular systole. François Franck ascribed it to the sudden lowering of the base of the heart at this phase of its cycle. Fredericq assigned its cause to the sudden diminution in volume of the auricle occurring when its walls return to their position of rest.

To Mackenzie[13] belongs the credit of having attracted the attention of clinicians to this third wave, which had escaped their notice until his excellent work, *The Study of the Pulse*, appeared.

The apparatus used to obtain the tracings shown in this article consists of two Marey tambours provided with long levers made of very thin, light straw, the writing points being short and thin strips of aluminum. The pulsations were taken up by means of Mackenzie's capsule, or by means of a small glass funnel, according as the one or the other gave the better results. So far as the venous pulsation is concerned, Mackenzie's capsule with a flat side which can be laid against the clavicle, is best adapted for the purpose. Needless to say, the rubber tubes leading from the receivers (capsules or glass funnels) to the tambours were exactly of the same length. Especial care was also taken before each observation to see that the writing points fell exactly on the same perpendicular line. The kymograph, on which the records were made, is the latest model of the Harvard kymograph, the speed of which can readily be varied to suit each particular case. The time record was obtained by means of a Page vibrator, the reed of which vibrated fifty times a second.

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[11] Mouvements des veines du cou en rapport, etc. Gaz. hebd. med. et chir., Mars et Avril, 1882. Nouvelles recherches sur un cas d'ectopie cardiaque pour servir a l'étude du pouls jugulaire, Arch. de phys., 1889, i, p. 70.

[12] Sur le pouls veineux physiologique, Travaux du laboratoire, 1889 to 1890, t. iii, p. 85. La pulsation du coeur ches le chien. Arch. de biologie, 1890, x.

[13] The Significance of the Venous Pulse. Edinb. Med. Jour., 1894. The Study of the Pulse and Movements of the Heart, London, Macmillan, 1903. The Interpretation of the Pulsations in the Jugular Veins. AMER. JOUR. MED. SCI. 1907, cxxxiv, 12.

The superiority of such an arrangement is obvious when one compares the results here presented with the tracings taken with even the best of the small sphygmographs. The clockwork of the latter instruments is often too slow to permit of an accurate estimation of slight differences of synchronism between two curves, a point which is not without importance, as we shall see later.

The venous pulsation was taken from the point at the root of the neck which gave the best excursion of the lever. In the great majority of instances this point is to be found on the right side, although in a certain number of cases the venous pulse is more marked on the left side. The best pulsating point is often over the position of the jugular bulb, but it is by no means rare that a better jugular pulse can be recorded when the receiver is placed over the lower part of the external jugular vein. Finally the patient was placed in the recumbent posture, for in many cases the jugular pulse disappears when the patient assumes a posture in which the trunk is erect. As dyspnœa is a disturbing factor, whenever this was present the patient was asked to breathe as quietly as possible.

For the purpose of timing the various events in the complete cycle of a jugular pulsation, a simultaneous record of an arterial pulse or of the heart beat is indispensable. The carotid pulse was, therefore, taken simultaneously with the jugular pulse, and in all those cases in which it was possible a record of the heart beat was also taken simultaneously with the jugular pulsation. In order to establish points of synchronism in the two curves, ordinates were drawn with the levers while they were still in the position they occupied during the time the tracing was being taken.

In Fig. 1 the complete cycle of a jugular pulsation is seen to consist of three positive waves, the first two of which, *a* and *s*, are relatively of short duration and have a sharp summit, while the third wave, *v*, is of longer duration and has a broad summit divided by a notch so that it presents a bifid appearance. These positive waves are separated by three depressions, or negative waves. The first two (2 and *Af*) are abrupt and occupy little time, while the third (*Vf*) situated between wave *v* and the next jugular pulsation, is more gradual in its descent, is deeper, and occupies a longer time in the cycle.

The first positive wave, *a*, is presystolic in time and is invariably interpreted as being due to the contraction of the auricle. The mechanism of its causation, however, admits of several interpretations. It may be due to the slowing or arrest of the venous flow with a resulting damming of the blood and increase in the diameter of the veins, or to the suddenness with which the blood stream is stopped, this giving rise to a wave travelling in a reverse direction. It may also be due to the propagation of a wave of regurgitation from the auricle.

Certain investigators, as Fredericq and Nuel,[14] do not believe that any regurgitation occurs in the great veins during auricular systole, under physiological conditions. On the other hand, Chauveau and Faivre[15] state, that in the horse there is normally a slight regurgitation of blood into the great veins during the contraction of the auricle. Keith[16] advances the opinion that the mouth of the superior vena cava is normally closed during the systole of the auricle by the contraction of a muscle band, the tinea terminalis, which performs the function of the venous valves found in the hearts of reptiles and batrachians. Should the auricle become engorged, however, the relaxation of this muscle band would allow of regurgitation into the veins, and Mackenzie thinks that it is easily rendered incompetent by changes affecting the tonicity of the auricle.

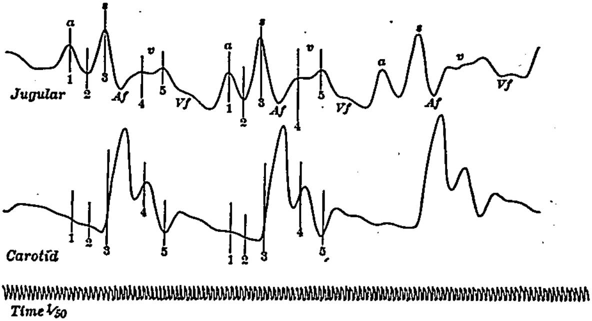

FIG. 1.—Simultaneous tracings of the jugular and the carotid pulses. From a case of mitral insufficiency and stenosis with perfect compensation. (Was admitted in the hospital for hydrops of the gall-bladder.) *a*, auricular wave; *s*, systolic wave; *v*, ventricular wave; *Af*, auricular filling; *Vf*, ventricular filling. The ordinates having the same numbers mark synchronous points on the curves. The letters and numbers have the same significance in all the tracings.

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[14] Elements de physiologie humaine, Gand, 1893.

[15] Nouvelles recherches experimentales sur les mouvements et les bruits normaux du coeur, envisagés au point de vue de la physiologie médicale. Gaz. med. de Paris, 1856, iii série, t. xi, p. 406.

[16] The Evolution and Action of Certain Muscular Structures of the Heart. Lancet, February 27, 1904 and March 5, 1904.

It is probable, however, that in the normal state the main factor in the formation of this wave is to be found in the slowing of the venous current, and the resulting accumulation of the blood in the veins, and, possibly, also in the production of a centrifugal wave originating through the sudden arrest of the inflowing blood.

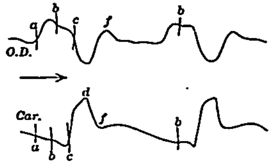

FIG. 2.—Simultaneous tracings of the intra-auricular pressure (*O.D.*) and of the pressure in the carotid artery (*Car.*) of a dog (morphine anesthesia). (Fredericq.) *a b*, auricular systole; *b c*, beginning of ventricular systole and projection of the auriculo-ventricular valves toward the auricle; *c d f*, outflow time; *f*, closure of semilunar valves (dicrotic notch).

The negative wave seen between *a* and *s* is easy of explanation. It is due to the collapse of the veins occurring with the resumption of the flow of the blood consequent on the relaxation of the auricle. This fall in the curve is, however, quickly interrupted by the positive wave, *s*, the interpretation of which has given rise to a great deal of controversy. It will be noticed that it is systolic in time, more correctly protosystolic, for it is normally synchronous with the beginning of ventricular systole. It reaches its height in the interval comprised between the beginning of the contraction of the ventricle and the opening of the semilunar valves, in other words, in that part of the cardiac cycle designated by the French as "systole préparatoire," and by the Germans as "Verschlusszeit" (Figs. 2, 3, and 4).

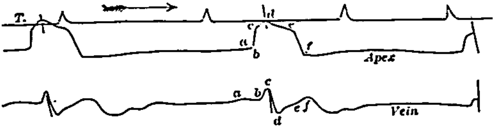

FIG. 3.—Simultaneous tracings of the apex beat and of the jugular pulse obtained from a large dog (morphine anesthesia). (Fredericq.) *a b*, auricular systole; *b c*, beginning of ventricular systole and projection of the auriculo-ventricular valves toward the auricle; *c d e*, (on apex tracing) systolic plateau; *e*, closure of semilunar valves; *e f*, relaxation of ventricles; *T*, time in seconds.

This wave was formerly considered abnormal and as being due to tricuspid regurgitation. This has been shown to be incorrect, as the jugular veins of normal animals exhibit it. Several explanations have been offered concerning its mode of production. Friedreich attributed it to the impact of the aorta against the superior vena cava. As already stated, François Franck believed it to originate in the sudden closure of the tricuspid valve, while Fredericq thought it was due to the projection of these valves toward the cavity of the auricle. Gerhardt[17] does not think that this latter explana-

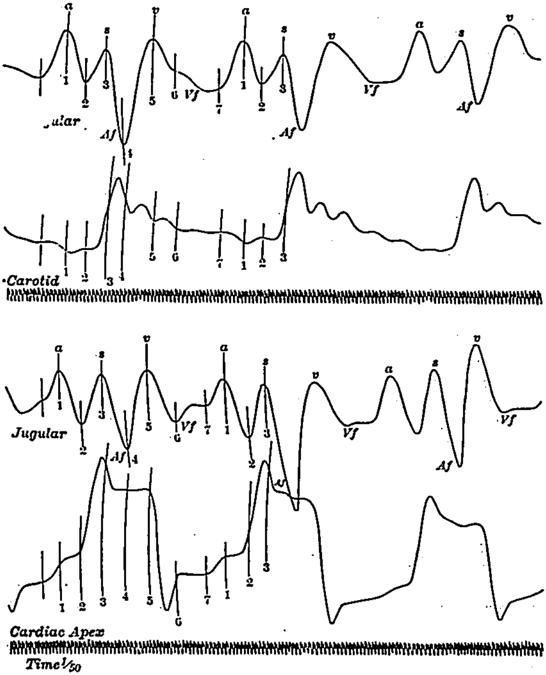

FIG. 4.—Simultaneous tracings of the jugular and the carotid pulses and of the jugular pulse and apex beat. Note that the upstroke of wave s coincides with the upstroke of the ventricular contraction (ordinate 2). Accordingly wave s precedes the carotid pulse by about 5/100 second. Wave v in this case is single. (Case of aortic regurgitation and relative mitral insufficiency with failing compensation.)

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[17] Klin. Untersuchungen über Venenpulsationen. Arch. f. exp. Path. u. Pharm., xxxiv, 402. Einige Beobachtungen am Venenpulse, Arch. f exp. Path. u. Pharm., 1902, xlvii, 250.

tion is warranted. He considers that the auriculoventricular opening is so tightly closed by the sphincter action of the ring of muscle around it, that as a result the valve leaflets are kept almost completely in apposition and that therefore they could not be pushed toward the auricular cavity by the sudden rise in ventricular pressure due to the contraction of the ventricle. He accordingly prefers Friedreich's explanation that the arteries coursing alongside the veins animate them with their own pulsation. Mackenzie adopted a similar interpretation, and he attributes the production of this wave as seen in the jugular pulse to the communicated impact of the neighboring carotid artery; hence the name he gave to it of "carotid wave." Mackenzie has advanced a number of arguments in support of his view. It is not within the scope of this paper to take them and discuss them seriatim. Those interested in the subject will finds the necessary information in papers by Morrow[18] and by Bard.[19]

No one will dispute the fact, however, that the pulsation of a contiguous artery may contribute to the formation of this wave. However, I do not believe that it arises entirely from this source, but rather that it is independent of it, and that all the pulsation of a neighboring artery can do is to add itself to it, or vitiate it. Bard states that he obtained this wave from a vein in front of the clavicle and that it occurs in those cases in which the carotid is pulseless, provided the ventricles do not fail to contract. Animal experimentation has made this point clear. Fredericq, Morrow, Cushny and Grosh[20] and others have shown that this wave can be obtained when methods of recording the jugular pulse are used that do not involve pressure over the carotid artery, as well as when the vein is carefully dissected away from the artery, or when the carotid is clamped close to the aorta. The objection can be raised, however, that these methods do not eliminate the influence of the aorta on the vena cava. An examination of the curves of intra-auricular pressure obtained by Marey, Fredericq (Fig. 2), Porter[21] and others shows the presence of this wave in the position mentioned above. Porter ascertained that when the ventricle is inhibited by stimulation of the vagus, this wave does not appear in the curve; he therefore concluded that it is due to the pushing of the auriculoventricular diaphragm toward the auricular cavity. The venous tracings illustrating this article show a striking resemblance to the curves of intra-auricular pressure given by Porter in his valuable paper.

That wave *s* is really independent of any arterial pulsation is shown by the fact that, in the majority of cases, it precedes the systolic line of ascent of the carotid pulse. So far as I am aware, Bard was the first to direct our attention to this point. An examination of the tracings shown here will reveal the truth of this assertion. In order to demonstrate this the tracings must be taken on a kymograph revolving at a sufficiently rapid rate to bring into evidence little

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[18] The Venous Pulse. Brit. Med. Jour., 1907, 777. The Various Forms of the Negative or Physiological Venous Pulse, ibid., 1906, 1807.

[19] De l'enregistrement graphique du pouls veineux des jugulaires chez l'homme. Jour. phys. et path. gener., 1906, t. VIII. Des divers détails du pouls veineux des jugulaires chez l'homme, ibid.

[20] The Venous Pulse. Jour. Amer. Med. Assoc., No. 15, xlix, 1254.

[21] Researches on the Filling of the Heart. Jour. Physiol., Cambridge, 1892, xiii, 513 to 553.

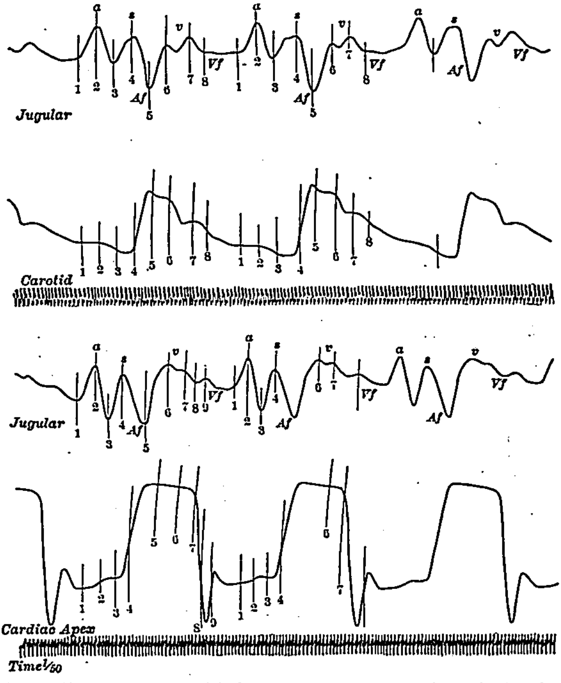

FIG. 5.—Simultaneous tracings of the jugular and the carotid pulses and of the jugular pulse and apex beat. From the same patient as Fig. 4 during a period of improvement.

differences of synchronism in the curves, and synchronous points should be marked with the levers in the manner already described, or by any other accurate method. The time elapsing between the beginning of wave *s* and the beginning of the carotid pulse varies in accordance with the degree of completion of the line of descent of the auricular wave, the rapidity with which the ventricle responds to the stimulus coming from the auricle as well as the rapidity and ease with which the ventricle overcomes the pulmonic pressure. This is well exemplified in Fig. 6 taken from a case of mitral stenosis

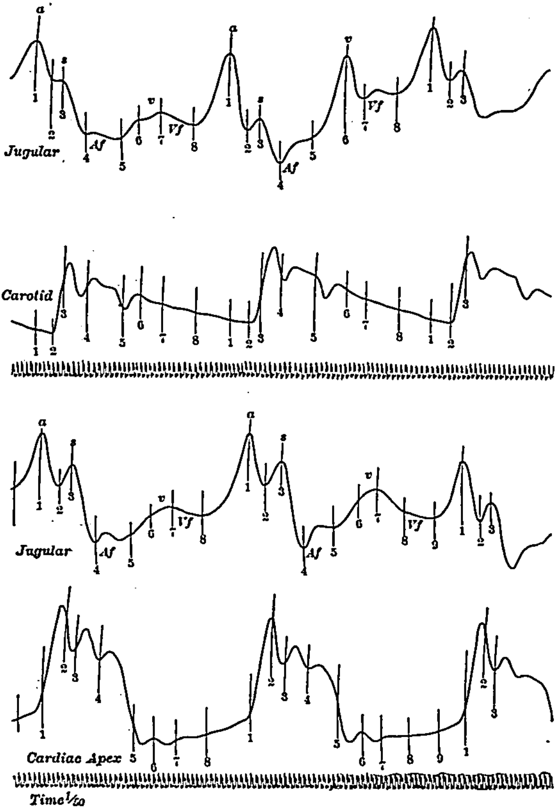

FIG. 6.—Simultaneous tracings of the jugular and the carotid pulses and of the jugular pulse and apex beat. Wave *v* occurs later in the cycle than in the other tracings. With the exception of the middle group, wave *s* begins with the carotid pulse. An examination of the lower tracings show that the ventricle responds quickly to the auricular impulse; wave *s* therefore occurs on the descending limb of wave *a*. (Case of mitral stenosis and regurgitation with beginning failure of compensation.)

in a fair state of compensation. According to Bard the difference in time between the appearance of this wave and the carotid pulsation, varies from $\frac{1}{100}$ to $\frac{2}{100}$ of a second. Until the present time, most of the tracings I have been able to obtain show somewhat higher figures, $\frac{4}{100}$ to $\frac{6}{100}$ of a second; in a few instances, however, Bard's figures were confirmed. In the middle group of Fig. 6, the time interval between ordinate 2 and the beginning of the carotid pulse is $\frac{2}{100}$ of a second.

Following the protosystolic rise *s*, a sudden fall occurs in the tracing producing the negative wave *Af*. This wave is generally attributed to the rapid emptying of the veins which takes place during the diastole of the auricle. The negative pressure in the auricle is due, not only to the dilatation of its valls, but in great part also to the action of the contracting ventricle. Chauveau and Marey[22] observed that the auriculoventricular septum is displaced toward the apex during the systole of the ventricle, this having for effect an enlargement of the auricular cavity. Porter offered another explanation of the production of a negative pressure in the auricle based upon the investigations of Purkinje, Nega, and Roy and Adami. He believes that it is due to the contraction of the papillary muscles pulling the auriculoventricular valves toward the ventricle.

Some investigators have thought that this negative wave is caused by changes in intrathoracic pressure coincident with the change in the volume of the heart occurring during ventricular systole. Gottwalt[23] showed this conception to be incorrect, inasmuch as this negative wave persists even though the chest be opened.

This wave, then, is indicative of the filling of the auricle, and is the product of several factors: the expansion of the auricle, the downward movement of the auriculoventricular septum, and the pull exerted by the papillary muscles on the auriculoventricular valve leaflets. It begins with the opening of the semilunar valves and lasts until toward the end of ventricular systole, when the third positive wave makes its appearance.

The normal position of the third positive wave usually called ventricular wave, *v*, is toward the end of ventricular systole. However, different observers do not agree to this, and some, like Gerhart, believe it to occur during ventricular diastole. The fact is that the relation of this wave to the cardiac cycle is not perfectly constant, and that pathological conditions within the heart seem to influence both its position and its form. It is not the purpose of this paper to discuss the pathological modifications of the venous pulse; I shall, therefore, consider it only as it occurs normally in the jugular veins of animals, and in those human beings who exhibit venous pulsations either without apparent pathological cause or as a result of some slight venous embarrassment.

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[22] Appareils et expériences cardiographiques, Mém. acad. med., Paris, 1863, t. xxvi, 313.

[23] Der normale Venenpuls. Arch. f. die ges. Physiol., 1881, xxv, 1.

Various explanations have been offered concerning the interpretation of this wave. Some, like Rigel, think that it is due to the communicated shock of the arterial dicrotic expansion; others, among whom is François Franck, see in it the effect of changes in intrathoracic pressure owing to the diastolic enlargement of the ventricle. But it has been shown that opening the thorax in animals does not affect the production of this wave. Mackenzie and others believe that it is due to the filling of the auricle and the consequent accumulation of the blood in the veins. Mackenzie, however, associates it in some way or other with tricuspid regurgitation.

While it is in all probability true that tricuspid regurgitation affects the *v* wave, it is none the less true also that it occurs in cases in which there is no regurgitation. It is found as a constant wave in the jugular pulse of animals, as well as in the curve of intra-auricular pressure. Moreover, the mechanism of its production is to be sought in the movements of the ventricle. Fredericq, Gerhart, Wenckebach, and others have ascribed it to the swinging back of the auriculoventricular septum. Morrow does not believe that the action of the ventricle is necessary for the formation of this wave, but Cushny and Grosh have shown that the suspension of the ventricular contractions is attended with the disappearance of this wave from the tracing.

It was stated above that two of the factors contributing to the formation of the negative wave *Af* are, on the one hand, the displacement of the auriculoventricular septum toward the apex, and, on the other hand, the pull of the papillary muscles on the tricuspid leaflets.

The relaxation of these papillary muscles and the return of the auriculoventricular septum to its former position decrease the cavity of the auricle, and, as the latter is filled, this encroachment upon its capacity has for effect an arrest of the flow and a damming of the blood in the veins. These two movements, however, are not synchronous, and, according to the researches of Roy and Adami,[24] the papillary muscles relax shortly before the beginning of ventricular diastole, hence before the return of the auriculoventricular septum to its position of rest. This relaxation occurs while the intra-ventricular pressure is still much higher than the intra-auricular pressure. We should, therefore, expect these two separate movements to be reflected in the wave under consideration. That this may be as here explained is seen by the constancy with which a notch is present on the summit of wave *v*, thus showing it to be made up of two lesser waves.

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[24] Heart Beat and Pulse Wave. Practitioner, London, 1890, xliv, 81, 161, 241, 347, 412; xlv, 20.

The variations in the form and in the position of this wave in the cardiac cycle are to be sought in pathological factors which may mask or annul the effect of the movements just described.

The third negative wave in the jugular pulse, *Vf*, occurs at the beginning of ventricular diastole, and is due to the opening of the tricuspid valve; the auricular blood entering the relaxing ventricle, the flow of the blood in the veins is accelerated, and the latter naturally decrease in diameter.

In almost all tracings taken from normal animals, either from the jugular vein or from within the auricle, this wave is more shallow than the negative wave *Af* (auricular filling). Although the tracing of Fig. 5 was taken from a pathological case of aortic and mitral regurgitation, what may be taken as the normal ratio of the depth of these two waves is well shown there.

It seems at first strange that this negative wave should be so little marked, but if it be remembered that the base of the ventricle may still be continuing its upward movement, though perhaps more slowly, it will be easily understood how this would counteract the effect of the aspirating action of the ventricle by gradually decreasing the capacity of the auricle.

A slight positive wave is very constantly present at or near the trough of this negative wave. It is in all probability a wave of stasis due to the entrance of blood into the veins from the periphery.

A moment's consideration will make it apparent that the study of the venous pulse by the graphic method is destined to give valuable information concerning the pathological function of the heart, for it can reasonably be inferred from what has been said, that it mirrors, more faithfully than the arterial pulse, the events occurring within the heart. Already it has proved invaluable in the study of heart-block, and in the study of the various forms of arrhythmia.

CONCLUSIONS. The physiological or so-called negative systolic venous pulse consists of three positive and three negative waves bearing a more or less definite relation to the events of the cardiac cycle and having their origin in the various movements of the chambers and structures of the right heart. The first positive wave (*a*) is presystolic in time and is due to the contraction of the auricle causing a slowing of the venous current and producing a centrifugal wave through a sudden arrest of the inflowing blood. The second positive wave (*s*) is protosystolic in time and originates in the sudden projection of the tricuspid valve into the cavity of the auricle during the quick, incipient rise in intraventricular pressure occurring in the protosystolic period. The third positive wave (*v*) occurs toward the end of ventricular systole. It consists of two lesser waves separated by a shallow notch. The factors entering into its formation are the relaxation of the papillary muscles at a time when the intraventricular is still higher than the intra-auricular pressure, this resulting in an upward movement of the tricuspid leaflets, and to the return of the auriculoventricular septum to its position of rest.

The first negative wave (between positive wave *a* and *s*) is due to the relaxing auricle. The second negative wave (*Af*) occurs during the diastole of the auricle. It is due to the dilatation of its walls, to the displacement of the auriculoventricular septum toward the apex occurring at the time of ventricular systole, and to the pull of the papillary muscles on the tricuspid valve leaflets. The third negative wave (*Vf*) appears during ventricular diastole and in the common pause of the heart chambers. Its cause is found in the passage of the blood from the auricle into the ventricle. It is somewhat modified, possibly by the continued ascent of the auriculoventricular septum and by a wave of stasis due to the accumulation of blood coming from the periphery.

I wish here to thank Professors A. P. Brubaker and W. M. L. Coplin for their encouragement and advice, and Professors J. C. Wilson and H. A. Hare from whose wards the necessary material was obtained.

FATIGUE IN SCHOOL CHILDREN AS TESTED BY THE ERGOGRAPH.

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The considerable number of children seen in dispensary service, particularly late in the winter and in the spring, suffering from nervous disorders, and the fact that these children are usually conscientious children who are making a desperate effort for a good showing at school, naturally leads one to suspect that school work is doing, at least some of the children, considerable harm. This feeling, I believe, is also shared by many school teachers.

Being interested in the physical welfare of the children in a large private school in New York, my attention was attracted to the work with the ergograph done under the direction of Dr. W. S. Christopher in the Chicago public schools during the years 1898, 1899, and 1900. Dr. Christopher was placed in charge of a department of child study, and undertook to apply various tests to the children so that each child might be so placed in school as best to acquire knowledge without physical harm. He tested the sight, hearing, lung capacity, etc. Among the various tests he applied to the children was a test of muscular activity, determined by the ergograph. The ergograph, which aims to use only one muscle and to exercise it until fatigue is produced, shows by comparative tests muscular fatigue. Dr. Christopher tested the children during various periods of the school day, and found in a considerable number of experiments that there is a diminution in the physical activity as shown by the ergograph, commencing within an hour of the beginning of the school day and proceeding until the noon recess, after which about the same activity was shown as at nine in the morning, but it fell off again during the afternoon session. The report of 1899-1900 notes one exception to this rule, and that was in four boys, who, on questioning, stated "that their teacher had been called from school and that they had had no regular work, but had been sent to another room but employed themselves, as they said, in having a good time."

The amount of deterioration in muscular activity during the school day, as shown in these children, was on the average during the first experiments from 20 to 25 per cent. A later average of 1127 records of one school showed a morning loss of 16 per cent., and an afternoon loss of only 4 per cent. This slighter evidence of fatigue, in the afternoon might be attributed to the fact that the most difficult school work is now arranged in the morning curriculum.

These very valuable observations by Dr. Christopher were published, but, so far as I know, no practical application was made of them; that is, no attempt was made to work out a school day which might reduce or eliminate fatigue, for it has been well demonstrated that fatigue, as shown by the ergograph, is very marked fatigue, for after slight fatigue a better tracing may be obtained than with no fatigue.

It seemed to me probable that by the use of more frequent recesses such fatigue might be eliminated and a school day elaborated, which would be free from constant marked fatigue and would keep the children in a condition more favorable to the acquisition of knowledge and without physical harm.

My first efforts to measure fatigue in school children were undertaken at the Charlton School three years ago. Two tests for fatigue were then used: that suggested by C. Ward Crampton, of New York, which consists in obtaining the difference between the blood pressure of a child standing and in a horizontal position, and the rapidity of the heart action in a child standing and in a horizontal position. This test was used in combination with a test by an ergograph devised by Dr. Thomas A. Storey and kindly lent to me by Professor Lombard, of Ann Arbor, in which the abductor indicis was used; each movement of the index finger toward the thumb raising a weight and making a tracing on a kymograph. Throughout this work I have been much indebted to Professor F. S. Lee, of the department of physiology of the College of Physicians and Surgeons, of Columbia University, for his wise advice and for the laboratory facilities and laboratory apparatus put at my disposal.

With these two tests I used children who were first required to concentrate in their studies; that is, children of about seven years. Absolutely no appreciable evidence of fatigue was obtained from the

Crampton test of blood pressure and pulse rapidity, and no general fatigue such as that described by Dr. Christopher was obtained by the use of this ergograph. Although slight fatigue was usually shown by some children, no persistent marked fatigue was ever found in any class of children.

The following year I continued the same experiments with the same apparatus in older children at the Charlton School with similar results. One ambitious and conscientious girl of high standing, with an acute mind, who was anemic and poorly nourished, showed constant fatigue after her morning work. Some others showed occasional fatigue, others none.

Having worked for two years with very little result in this school I arranged during the past winter to conduct work in the public schools, and through the courtesy of Health Commissioner Darlington and Superintendent Maxwell of the Board of Education, and Superintendent Boyle, of Public School 69, I was enabled to obtain the facilities that I needed. I used for this purpose at first girls eleven years old, taking first a group of five whom their teacher thought were fatigued at the end of the school day. These children, who were apparently showing some fatigue, showed no marked fatigue such as Dr. Christopher observed in Chicago. Other groups of five children of the same age that were afterward taken gave similar results. I therefore concluded that further work with this ergograph was useless, either in order to confirm or obtain conclusions on the work done by Dr. Christopher.

I then procured a Mosso ergograph (Fig. 1), similar to the one used in Chicago, which was kindly lent to me by Professor Howell of Johns Hopkins University, and to this was attached an adder. This Mosso ergograph uses the flexor of the middle finger. It was arranged so that each flexion of the middle finger raises a weight, makes a tracing on the kymograph as well as registering the amount of work done on the adder. In the use of this apparatus the method followed in Chicago was adopted, each child pulling 7 per cent. of his weight thirty times a minute for one and one-half minutes, thus giving each child about forty-five pulls.

With this apparatus I have tested a considerable number of children in a New York public school and have obtained results exactly similar to those obtained with the other form of ergograph which I previously used. While some children show fatigue in their tracings after an interval of school work, other children will make better tracings than at the beginning.

Twenty children tested 9.30 A.M. showed an average of 264 kg. cm. of work; at 11.30, 286; at 1.30, 272, and at 3.30, 250. This shows a slight increase in muscular activity at 11.30 over 9.30 and again a slight diminution at 2.30 as against 1.30 (Fig. 2).

An average of 36 observations at 9.30 and 11.30 on both boys and girls from ten to fourteen years of age, gave an average result of 278 kg. cm. at 9.30 and 282 kg. cm. at 11.30, a slight increase in the amount done at 11.30 over 9.30. As some of these children were

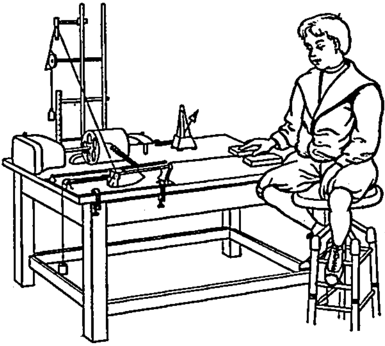

Fig. 1.—A child being tested with the Mosso ergograph.

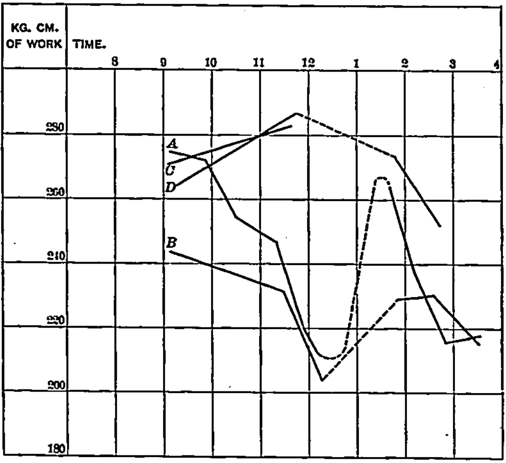

Fig. 2.—Influence of school work on physical activity. *A*, 8 Chicago public school children; *B*, 1127 Chicago public school children; *C*, 36 New York public school children; *D*, 20 New York public school children.

using the instrument for the first time on this day an estimate of the work done by children who had repeatedly used the ergograph might help to eliminate this possible error. Eleven children familiar with the instrument did an average of 204 kg. cm. of work at 9.30 and 256 at 11.30, so that there seems no way that we can use these figures to obtain results similar to those obtained by Dr. Christopher. This may be accounted for by a difference in the school curriculum. It is possible that the duties required of the pupils in Chicago are more difficult and that the sessions possess less opportunities for recesses. This would, of course, alter the results. As I have been unable to obtain the curriculum used in Chicago I cannot compare it with that used in New York.

As a conclusion, I might say that the Crampton test for fatigue has given negative results as applied to school children in New York; that the Storey ergograph as used for the detection of fatigue in school children has given negative results and is unsuitable on account of the use of accessory muscles when the abductor indicis becomes fatigued; that the Mosso ergograph combined with an adder as used in the Chicago public schools is the more reliable instrument for this purpose, but in my hands fails to give evidence of marked fatigue of the pupils during the school day in a private and also in a public school of New York City; and that in occasional children either form of ergograph will give evidence of fatigue.

The ergograph, so far as my work indicates, is of no value in modifying the school day in the public schools of New York for the average student, but might be shown to be of value in lightening and controlling the work for those pupils who give evidence of marked fatigue during the school day.

THE DIAGNOSTIC VALUE OF CUTANEOUS HYPERALGESIA (HEAD'S ZONES) IN ABDOMINAL DISEASE.

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Although fifteen years have passed since Henry Head first published his discovery of the association of increased sensitiveness of the skin with visceral disease, the literature of the subject has remained very small. Lange is said to have written about skin sensitiveness in visceral disease as early as 1871. Ross referred to the subject in 1888, but Head was the first to make a careful study of the phenomena. Notwithstanding the importance of Head's publication, it seems to have attracted little attention outside of England. The papers of Mackenzie, Thorburn, Thane, and others, in England, and of Petren and Faber, on the Continent, are the only ones of importance that have appeared. In most of the text-books the subject has been dismissed with a few lines.

During the last two years we have made a large number of examinations in the wards of Mt. Sinai Hospital, to determine the presence or absence of areas of hyperalgesia in abdominal affections. The majority of the patients were admitted to the surgical service of Dr. Lilienthal, and were sooner or later submitted to operation, so that the results of our observations were verified by the operative findings. In the following paper are given the results of our studies, arranged under the following headings:

I. Description of Head's zones.
II. Methods to be used in testing for cutaneous sensitiveness.
III. The appearance and disappearance of zones of hyperalgesia in abdominal disease.
IV. The general significance of localized areas of cutaneous hyperalgesia in visceral disease.
V. General consideration of the subject of hyperalgesic zones in abdominal disease.
VI. Head zones in affections of the various viscera.
VII. Conclusions.

I. DESCRIPTION OF HEAD'S ZONES.

In 1893 and later, Henry Head described certain definite and constant areas of cutaneous tenderness associated with diseases of the different viscera. He found that in many visceral affections, if the sensitiveness of the skin was tested by running a pin point over the cutaneous surface, there could be shown to exist areas over which there was a more or less marked hypersensitiveness to pain. As a result of painstaking investigations extending over many years, he concluded that these areas were constant and distinct; they could be mapped out on the surface of the skin, and, when present, were an almost infallible sign of an affection of the organ to which they corresponded. The skin tenderness was very superficial—quite different from tenderness on deep pressure—and extended over definite areas which never overlapped one another. Each area or "zone of hyperalgesia," as he called it, had a "maximum" region, which often corresponded to the location of the pain complained of by the patient, and coincided with the areas marked out by attacks of herpes zoster. Head found, also, that these areas were hypersensitive to heat and cold, but not to touch. He concluded that there is